THE EFFECT OF EXPERIMENTAL LOW BACK PAIN ON LUMBAR MUSCLE ACTIVITY IN PEOPLE WITH A HISTORY OF CLINICAL LOW BACK PAIN: A MFMRI STUDY

Jessica Van Oosterwijck*, Barbara Cagnie*, Roseline D'hooge*, Yves De Deene #, Geert Crombez•, Guy Vanderstraeten*, Thierry Parlevliet⅄, L.ieven Danneels*

Ghent University, Belgium: * Department of Rehabilitation Sciences and Physiotherapy, • Department of Experimental Clinical and Health Psychology
Ghent University Hospital, Belgium: # Department of Radiotherapy and Experimental Cancer Research, ⅄ Department of Physical Medicine and Orthopedic Surgery

BACKGROUND AND AIM

Experimental pain models have been used to study the causal effect of peripheral nociception on motor output. Previous studies have demonstrated altered muscle behavior during experimental low back pain (LBP) in healthy people. Although comparable observations have been made in clinical LBP, changes in motor output in relation to clinical LBP do not only depend upon peripheral nociceptive stimuli, but are the net resultant of a complex interaction at multiple levels along the sensory, central and motor nervous system.

In people with a history of clinical recurrent LBP, structural and functional alterations have been observed at several peripheral/central levels of the sensorimotor pathway. These alterations might interact with the way the sensorimotor system responds to pain, and current study aimed at examining this assumption.

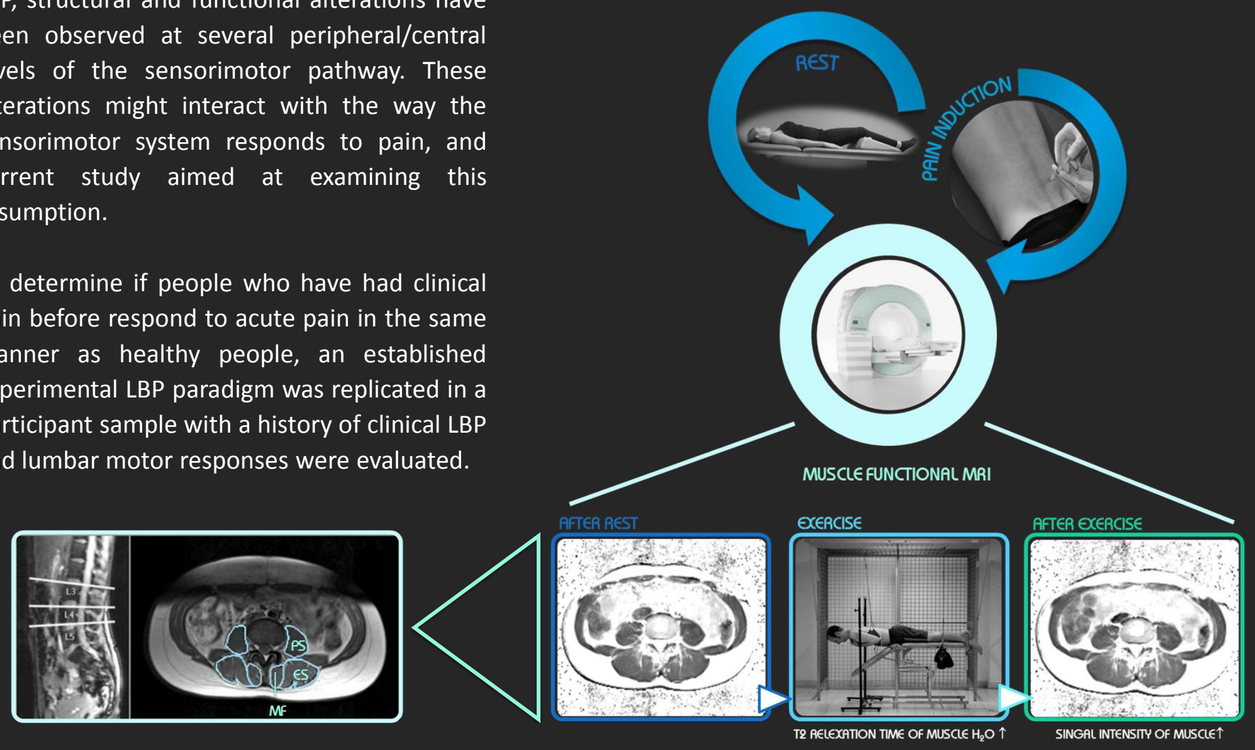
To determine if people who have had clinical pain before respond to acute pain in the same manner as healthy people, an established experimental LBP paradigm was replicated in a participant sample with a history of clinical LBP and lumbar motor responses were evaluated.

METHODS

The effect of an experimental pain paradigm on lumbar muscle activity was evaluated in 15 participants during remission of unilateral recurrent LBP. Participants their mean age was 37±12 years. The mean duration of LBP since first onset was 96±85 months and a mean frequency of 5±2 episodes per years was reported.

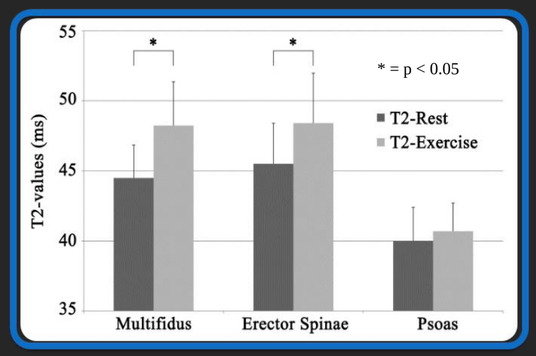
Quantitative T2-images (muscle functional MRI) were taken bilaterally of multifidus, erector spinae and psoas at the L3 upper/L4 upper and lower endplate during several conditions:

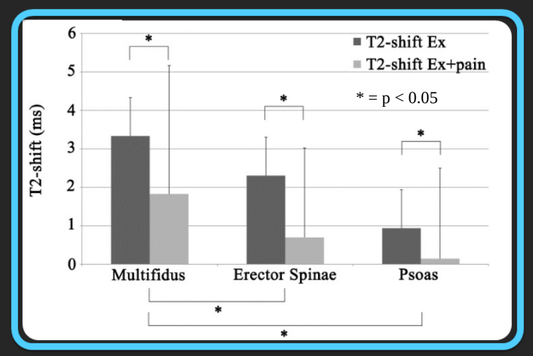
- at rest after 30 min of supine lying
- upon trunk-extension exercise (10 reps at 40%-1RM)
  - without pain
  - with experimental induced LBP at the clinical pain-side



RESULTS

T2-values were significantly higher in the exercise condition (without pain) compared to the resting condition for MF (p<.001) and ES (p=.003), but not for PS (p=.281).
T2-shift was significantly lower in the exercise-with-pain compared to the exercise-without-pain condition for all 3 muscles, on both painful and non-painful sides, and at multiple segmental levels (p=.038). Pain intensity and localization from experimental LBP were similar as during recalled clinical LBP episodes.





CONCLUSIONS

Administration of unilateral experimental LBP in people with a history of clinical recurrent LBP effected a generalized, widespread inhibitory response in lumbar muscle activity during trunk extension. This response was consistent with previously established inhibitory patterns in healthy people in response to experimental induced LBP, and appeared despite and in addition to the presence of pre-existing dysfunctions during remission of recurrent LBP. It is striking that similar inhibitory patterns in response to pain could be observed, despite the presence of pre-existing alterations in the lumbar musculature during remission of RLBP. These results suggest that motor output can modify along the course of RLBP., which encourages the need for further research to unravel the longitudinal course of muscle recruitment and the involved pathophysiological mechanisms during and after episodes of recurrent LBP.

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CONTACT DETAILS: jessica.vanoosterwijck@ugent.be